In the Specification:

Please amend the specification as shown:

Please delete the paragraph [0034] and replace it with the following paragraph:

[0034] The type I receptor, but not the type II, contains a characteristic SGSGSG sequence **(SEQ ID NO: 22)**, the "GS domain," immediately N-terminal to the kinase domain. The type II receptor phosphorylates multiple serine and threonine residues of the type I GS domain, thereby activating type I. Thus, the GS domain of the type I receptor serves as an important regulatory domain for TGF-beta signaling. As such, the GS domain of the type I receptor is a candidate for thioaptamer targeting, to facilitate study of its role in TGF-β signaling. For example, the immunophilin FKBP12 was shown to inhibit TGF-β signaling by binding to the unphosphorylated GS domain of type I receptor (Huse, et al., 1999), so that it cannot interact with its downstream target, the R-Smad proteins.

Please delete the paragraph [0044] and replace it with the following paragraph:

[0044] For a more complete understanding of the features and advantages of the present invention, reference is now made to the detailed description of the invention along with the accompanying figures and in which:

Figure 1 is a Clustal W (1.8) alignment of sequences of clones clones **(SEQ ID NOS 24-60, respectively, in order of appearance)** isolated during the selection of thioaptamer binding TGF-β1 (through 12th round of selection);

Figure 2 is a predicted secondary structures of highest affinity (to TGF-β1) thioapter of rounds 5,9,12 (thioaptamers T5_14 **(SEQ ID NO: 61)**, T9_5 **(SEQ ID NO: 62)**, T12_8 **(SEQ ID NO: 63)**);

Figures 3a, 3b, 3c and 3d are gels of electromobility shift assays of the initial library (3a) and of thioaptamer candidates T5_14, T9_5 and T9_22 (3b, 3c, 3d);

Figure 4a, 4b and 4c show models of a predicted molecular nature of three DNA bands in the corresponding portions of a gel; and

Figure 5 is an analysis of binding of T9_5, T9_22 and initial library to a TGF-β1 target protein.

Please delete the paragraph [0061] and replace it with the following paragraph:

[0061] Thiophosphate aptamers are capable of specifically and non-specifically binding to proteins. Importantly, the present inventors have observed that sulfurization of the phosphoryl oxygens of oligonucleotides often leads to their enhanced binding to numerous proteins. The dithioate agents, for instance, appear to inhibit viral polymerases at much lower concentrations than do the monothiophosphates, which in turn are better than the normal phosphates, with K_d's for single strand aptamers in the nM to sub-nM range for HIV-1 RT and NF-κB. For HIV-1 RT, dithioates bind 28-600 times more tightly than the normal aptamer oligonucleotide or the S-analogue. Sequence is also important, as demonstrated by the observation that a 14-nt dithioate based on the 3' terminal end of human $tRNA^{Lys}$ complementary to the HIV primer binding site is a more effective inhibitor (ID_{50} = 4.3 nM) than simply dithioate dC_{14} **(SEQ ID NO: 23)**(ID_{50} = 62 nM) by an order of magnitude.